Diet and risk of ischemic heart disease in India[1–3]

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ABSTRACT

Background: Ischemic heart disease (IHD) is a leading cause of death in India. Dietary changes could reduce risk, but few studies have addressed the association between diet and IHD risk in India.

Objective: The goal was to address the association between diet and IHD risk among Indians in New Delhi (northern India) and Bangalore (southern India).

Design: We collected data from 350 cases of acute myocardial infarction and 700 controls matched on the basis of age, sex, and hospital as part of a hospital-based case-control study in 8 hospitals. Long-term dietary intake was assessed by using food-frequency questionnaires developed for New Delhi and Bangalore. We used conditional logistic regression to control for the matching factors and other predictors of risk.

Results: We observed a significant and dose-dependent inverse association between vegetable intake and IHD risk. The inverse association was stronger for green leafy vegetables; in multivariate analysis, persons consuming a median of 3.5 servings/wk had a 67% lower relative risk (RR: 0.33; 95% CI: 0.17, 0.64; P for trend = 0.0001) than did those consuming 0.5 servings/wk. Controlling for other dietary covariates did not alter the association. Cereal intake was also associated with a lower risk. Use of mustard oil, which is rich in α-linolenic acid, was associated with a lower risk than was use of sunflower oil [for use in cooking: RR: 0.49 (95% CI: 0.24, 0.99); for use in frying, RR: 0.29 (95% CI: 0.13, 0.64)].

Conclusion: Diets rich in vegetables and use of mustard oil could contribute to the lower risk of IHD among Indians. *Am J Clin Nutr* 2004;79:582–92.

KEY WORDS Diet, nutrition, ischemic heart disease, India, foods, vegetables, green leafy vegetables, fats, oils

INTRODUCTION

Cardiovascular diseases in India cause 3 million deaths/y, accounting for 25% of all mortality (1). The World Health Organization predicts that deaths due to circulatory system diseases are projected to double between 1985 and 2015 (2-4). Moreover, research on Indian Asians living abroad indicates a 40% higher risk of ischemic heart disease (IHD) mortality than that for Europeans (5).

Dietary factors that may contribute to a high IHD risk in India include low intakes of vitamin B-6 and folate (6) and high intakes of *trans* fatty acids, which have been associated with risk in studies conducted in the West (7-14). In parts of India, *trans* fats from hydrogenated vegetable oil in the form of *vanaspati* are consumed in greater quantity than in the United States (10, 15). In contrast, in North India, the most commonly used oil in cooking is mustard oil. Mustard oil (*Brassica juncea*), like canola oil, is produced from rapeseed, a member of the crucifer family that is rich in α-linolenic acid (18:3n−3), which may reduce the risk of IHD (16-18). To address the relation between diet and IHD among Indians, we conducted a case-control study in 2 major Indian cities.

SUBJECTS AND METHODS

The study was approved by the relevant institutional review boards.

Study participants

Cases

Eligible cases were all patients aged 21-74 y hospitalized with a diagnosis of first incident acute myocardial infarction (MI) in 1 of 8 urban hospitals in New Delhi or Bangalore between 15 January 1999 (or later dates in 1999 for some of the hospitals) and either 21 December 1999 (New Delhi) or 16 January 2000 (Bangalore). The initial 3 participating hospitals were also part of an investigation of the Indian Council of Medical Research, so case subjects were selected according to Indian Council of Medical Research study criteria (19). Definite diagnosis of acute MI was based on clinical examination, electrocardiogram readings, and measurement of cardiac enzymes. Research assistants identified cases by visiting participating hospitals daily or biweekly and by consulting the physicians on duty. Patients were excluded if they had any previous history of MI or IHD (including bypass surgery, angina, or stroke) because such prior diagnoses may alter behaviors, including diet. We also excluded patients if they were pregnant, had a history of cancer, or had a chronic disease of the kidney, liver, gastrointestinal tract, or thyroid, because these conditions may have affected dietary intake. Patients who had an

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acute viral infection in the 4 wk before admission were also excluded, as were those who failed to provide informed consent. The patients were interviewed on average 2–5 d after admission. The eligibility criteria were met by 419 cases, and 350 were included in the study. The reasons for exclusion were death ($n = 25$) or discharge ($n = 23$) before the interviews could be completed, being too sick to be interviewed ($n = 13$), and not giving consent to participate ($n = 8$).

Controls

For each case subject, 2 control subjects matched by age (within 5 y), sex, and hospital were obtained from noncardiac outpatient clinics or inpatient wards. The same exclusion criteria used for cases were applied for control selection. We identified ≈707 eligible control subjects, of whom 7 declined to be interviewed. The controls were relatively healthy individuals with minor ailments or conditions and were enlisted from the following wards and clinics (% of total controls): eye (37%); ear, nose, and throat (21%); dermatology (14%); orthopedics (10%); surgery (7%); general medicine (7%); gynecology (3%); and other (< 1%).

Controls were selected by using predominantly 1 of 2 methods depending on the hospital. In the first method, research assistants were assigned to accompany a particular physician during an outpatient clinic, according to a weekly schedule of clinics and wards. At the end of each consultation, the physician or the physician's assistant invited the patient to speak with the research assistants about his or her lifestyle and diet. Patients matching the required age and sex profile and who were eligible according to study criteria were then informed of the study and asked to participate. In these situations, participation was 100%. In the second method, the research assistants independently identified control patients from clinics and wards. The assistants attempted to approach all individuals present during a particular outpatient clinic or in a specified ward. In large clinics, patients were screened for eligibility and invited to participate according to their queue number (highest number first). This method was used to prevent bias in the selection of controls. Overall participation was high, ≈99%. Basic demographic information was collected from all persons who were approached. If an individual fit the required age and sex profile and was eligible, the assistants briefly explained the study and asked whether the patient was willing to participate.

Data collection

Interviews were conducted in hospital wards or clinics by 1 of 4 research assistants and lasted ≈25 min. Informed consent was obtained from all study subjects. Research assistants collected data on socioeconomic status; smoking history; history of hypertension, diabetes, and hypercholesterolemia; family history of cardiovascular disease (including IHD, angina, MI, hypertension, diabetes, stroke, sudden death, and bypass surgery); dietary intake; types of fat or oils used in cooking; nutritional supplement use; and physical activity. Next, anthropometric measures (height, weight, and hip and waist circumferences) were obtained and body mass index (weight in kg divided by height in meters squared) and waist-to-hip ratio were calculated. Waist and hip measures were assessed by using a standardized tape measure, with waist measures taken at the midpoint between the costal margin and ileac crest and hip measures taken at the widest circumference.

Food-frequency questionnaires

Food-frequency questionnaires (FFQs) were used to obtain a measure of long-term dietary intake. We developed 2 separate FFQs, one for New Delhi and one for Bangalore, to take into account regional differences in food intake. The FFQs followed the format of the questionnaires developed at Harvard University, Department of Nutrition, except that the response categories were open-ended.

To determine the list of foods for the New Delhi FFQ, dietitians collected ≈100 24-h dietary recalls from subjects in outpatient clinics at the All India Institute of Medical Sciences in New Delhi, a population similar to the control group of the main case-control study. Nutritionists then assisted in constructing a list of food items that were important nutrient sources. The New Delhi FFQ was field-tested on 50 individuals, and several revisions were made before the FFQ was implemented in the study. The New Delhi FFQ had a food and beverage list of 149 items.

Nutrition researchers in Bangalore (St John's Medical College) used data from 180 dietary recalls and a comprehensive database of South Indian foods to develop a FFQ specific for their region. We made modifications to the food list and revised the format to develop the Bangalore FFQ, which ultimately contained 141 food and beverage items.

Research assistants asked the subjects to indicate the average frequency of consumption of each food item over the past 1-y period (for cases, before the MI event). For each item, an open-ended numerical response was entered into one of the following categories: per day, per week, per month, or per year. If the subject did not consume that item, the "never" box was marked. Information on all items in the food list was obtained. Research assistants showed subjects standard measures (bowls, glasses, etc) at the start of the interviews to ensure that responses corresponded with the average serving sizes listed. The subjects were asked whether certain fruit and vegetables were consumed in season or year-round and were then asked to provide the frequency of intake during that period. Data on the average seasonal availability (in months) of such fruit and vegetables were obtained from local wholesale vendors and agricultural institutes. These values were used to convert reported seasonal intakes into average intakes throughout the year.

Responses for foods items were converted to average number of servings per day, per week, or per month. Foods were categorized as follows: dairy foods ($n = 10$ food items), vegetables ($n = 21$), fruit ($n = 16$), breads and cereals ($n = 26$), beans ($n = 5$), meats ($n = 10$), fish ($n = 4$), mixed dishes ($n = 7$), and snacks and desserts ($n = 20$). (*See* **Appendix A** for a list of the foods in each group.) Intakes of all foods (servings per day or wk) within each group were summed to get servings per day, week, or month of each food group. Green leafy vegetables were assessed separately, as were potatoes (including yams), eggs, tea, coffee, and alcohol. The FFQs also assessed the amount of ghee (clarified butter), vanaspati (hydrogenated vegetable oil), and vegetable oil added to foods. Additionally, questionnaires included a section on dietary habits in which information on the specific types of fats or oils used in regular cooking and in deep frying foods was obtained. Data on special diets and vitamin and supplement usage were collected.

Physical activity levels were assessed by using a validated physical activity questionnaire specific for the Indian population that focused on occupational and other non-leisure-time activities in addition to leisure-time activities (20). The questionnaire was validated by comparing energy expenditure (determined by the questionnaire) with energy intake as measured by 24-h dietary recalls. Subjects were asked to report the average time spent at work and the frequency of activities related to leisure or recreation, household chores, and sedentary and daily activities. The questionnaire provided an estimate of overall energy expenditure per day and the energy expenditure of specific categories of activities (work, leisure-time exercise, hobbies, chores, and sedentary activities). Metabolic equivalent-minutes (MET-min), a measure of both intensity and duration of specific activities, were also derived.

Statistical analysis

IHD risk factors were compared between cases and control subjects by using *t* tests for matched data, signed-rank tests for continuous variables, and chi-square tests for categorical variables. To assess the potential for confounding, mean values or the prevalence of IHD risk factors across food group intake categories (average number of servings per day) among controls were examined. The relation between intakes of vegetables, *trans* fatty acids, and mustard oil and IHD risk was examined by conditional logistic regression, first with control for only the matching factors (age, sex, and hospital) and then with additional control for other potential risk factors. Analyses of food groups compared persons in increasing categories of intake (in average number of servings per day or week) with persons in the lowest category. Continuous covariates were assessed categorically to avoid assumptions of linear associations with the outcome and to minimize the effect of outlying values. The relation between types of fats or oils used in cooking and frying and IHD risk was also assessed in conditional logistic regression analysis with the reference group being users of sunflower oil. Risk according to use of ghee, vanaspati, mustard oil, peanut oil, sunflower oil, or safflower oil was assessed categorically, and a Bonferroni correction was used to adjust for multiple comparisons in which only P values < 0.01 were considered to be statistically significant. We examined whether the observed associations with intake of specific food groups, fats, and oils and risk of acute MI were modified by age, sex, cigarette and bidi smoking, body mass index, waist-to-hip ratio, physical activity, alcohol intake, education, city, or income. All analyses were conducted with STATISTICAL ANALYSIS SOFTWARE (version 8; SAS Institute Inc, Cary, NC).

RESULTS

We enrolled 350 cases and 700 controls, recruited equally from New Delhi and Bangalore. The subjects' mean ($\pm$ SD) age was 52 ± 11 y, and 12% of the subjects were women.

About 38% of the study participants followed vegetarian diets, consuming no meat, chicken, fish, or eggs. The percentage of subjects who were vegetarian yet consumed eggs was slightly higher (45%). The major contributors to total vegetable intake in our population were potatoes, salads (usually small portions of fresh tomatoes, cucumber, or onions), green leafy vegetables, gourds, okra, and carrots. Commonly eaten fruit were bananas, mangos, citrus fruit, papaya, and apples. The foods that contributed most to total cereal intake were roti (Indian flat bread made from wheat flour), rice, and sliced white bread.

The mean and percentage values of various cofactors among the cases and controls are presented in **Table 1**. In our study population, case subjects had significantly higher body mass indexes, waist-to-hip ratios, and alcohol intakes and a significantly higher prevalence of history of hypertension, high cholesterol, diabetes, and family history of IHD. However, cases had significantly lower intakes of green leafy vegetables and mustard oil and participated in less exercise than did control subjects.

The mean values of IHD risk factors across vegetable intake categories are presented in **Table 2**. Persons consuming higher amounts of vegetables tended to be of higher socioeconomic status than those who consumed less. They also had slightly higher body mass indexes and were less likely to smoke bidis.

The relative risk (RR) of IHD according to potential risk factors is presented in **Table 3**. In age- and sex-adjusted analyses and in multivariate-adjusted analyses, cigarette and bidi smoking, body mass index, waist-to-hip ratio, leisure-time physical exercise, family history of IHD, history of hypertension, and education were significant predictors of IHD in this population.

In conditional logistic regression analysis, increased total vegetable intake (not including potatoes) was significantly associated with a lower risk of IHD in the analysis adjusted for age, sex, and smoking (**Table 4**). The association was strengthened after further control for other risk factors. In comparison with persons consuming a median of 0.8 servings/d, persons consuming 3.5 servings of vegetables/d had an RR of 0.27 (95% CI: 0.11, 0.64; P for trend $= 0.006$). Further control for dietary covariates slightly attenuated the association (RR: 0.33; 95% CI: 0.13, 0.82; P for trend $= 0.006$).

Among the vegetables, the strongest associations were observed for green leafy vegetables, both in univariate and multivariate analysis. Persons consuming a median of 3.5 servings/wk had an RR of 0.33 (95% CI: 0.17, 0.64; P for trend $= 0.0001$) compared with those consuming 0.5 servings/wk. Further control for other vegetables did not substantially alter this estimate. In contrast, consumption of potatoes was not associated with risk.

Fruit intake was associated with an increase in risk (RR: 2.11; 95% CI: 1.03, 4.32; P for trend $= 0.06$), although this was not significant in the multivariate analysis, when comparing persons who consumed > 3 servings/d with those who consumed ≤ 1. Further control for dietary covariates strengthened this association (RR: 2.46; 95% CI: 1.15, 5.25; P for trend $= 0.03$).

No significant interaction was found between intakes of total vegetables or green leafy vegetables and age, sex, cigarette and bidi smoking, body mass index, waist-to-hip ratio, physical activity, alcohol intake, education, income, and city. Moreover, the associations were not altered after control for other dietary variables, including cereal intake and type of oil used in cooking and frying.

We also observed an inverse association between cereal intake and IHD risk in both univariate and multivariate-adjusted analyses, whereas beans and dairy foods were not significantly associated with risk of IHD in multivariate analyses (Table 3). Most of the apparent effect with cereal intake was attributable to consumption of roti (wheat flat bread) (data not shown). In multivariate analysis of roti intake (with additional control for other cereal foods), we found that persons consuming 9 servings/d had an RR of 0.38 (95% CI: 0.15, 0.92; P for trend $= 0.01$) compared with those with no intake. This association did not change with further adjustment for green leafy vegetables and use of mustard oil. No association was observed with rice intake.

TABLE 1
Distribution of ischemic heart disease (IHD) risk factors among case and control subjects

	Cases ($n = 350$)	Controls ($n = 700$)
Age (y)	52.4 ± 10.6[1]	51.6 ± 11.0[2]
BMI (kg/m^2)	23.8 ± 3.7	23.3 ± 4.0[3]
Waist-to-hip ratio	0.96 ± 0.07	0.94 ± 0.07[2]
Alcohol intake (servings/d)	0.28 ± 0.79	0.16 ± 0.72[3]
Physical activity (MET-min/d)[4]	66.29 ± 133.7	98.67 ± 161.7[5]
Total vegetable intake (servings/d)[6]	2.57 ± 1.18	2.67 ± 1.24
Green leafy vegetable intake (servings/d)	0.23 ± 0.27	0.27 ± 0.25[3]
Current cigarette smokers (%)	30.0	13.3[2]
Current bidi smokers (%)	25.4	11.4[2]
Family history of IHD (%)	41.7	31.1[2]
History of hypertension (%)	30.3	16.3[2]
History of high cholesterol (%)	2.6	0.6[2]
History of diabetes (%)	17.4	13.0
Use of mustard oil in cooking (%)	22	28[3]

[1] $\bar{x}$ ± SD (all such values).
[2,3,5] Significantly different from cases (t test for matched data, signed-rank test for continuous variables, and chi-square test for categorical variables): [2] $P \leq 0.0001$, [3] $P < 0.05$, [5] $P = 0.003$.
[4] MET, metabolic equivalent.
[6] Does not include potatoes.

Meat (including chicken, goat, beef, and pork) intake was associated with a significant reduction in risk compared with nonconsumption in multivariate analysis; however, this association was attenuated after further control for other food groups. Intakes of fish, eggs, tea, coffee, alcohol (**Table 5**), snacks and desserts, and mixed dishes were not significantly associated with risk. However, a suggestive trend of reduced risk was observed with fish intake (RR: 0.69; 95% CI: 0.46, 1.03). Further control for other dietary variables did not appreciably change the association. An association with vegetarianism was not observed.

Persons adding vanaspati (hydrogenated vegetable oil) to foods (**Table 6**) were at slightly higher risk (although not significantly so) for IHD than were those who did not, with an RR of 1.81 in multivariate analysis (95% CI: 0.99, 3.31). This association was attenuated with further control for other food groups.

Subjects were asked to report the type of oils or fat used in regular cooking and in deep-frying. Sunflower, mustard, and peanut (groundnut) oils were most commonly used in this population, with more persons using vanaspati for deep-frying than for regular cooking. Use of mustard oil was reported more frequently in New Delhi than in Bangalore and was significantly higher among controls than among cases. Compared with persons consuming sunflower oil, those using mustard oil for cooking had an RR of 0.44 for IHD in the age-, sex-, and smoking-adjusted analysis (**Table 7**). In multivariate analyses, use of mustard oil was associated with an RR of 0.49 (95% CI: 0.24, 0.99) compared with use of sunflower oil for cooking. Further control for other dietary factors including intakes of green leafy vegetables and cereals slightly strengthened the association. Similarly, persons using mustard oil for frying foods had a 71% lower risk (RR: 0.29; 95% CI: 0.13, 0.64) in multivariate analysis. The association was also slightly strengthened after further adjustment for dietary covariates. When compared with all other fats and oils, the inverse association with mustard oil remained. An association with vanaspati used in cooking or frying, a quantity greater than that consumed in discretionary vanaspati use, was not observed. No significant interaction was found between use of mustard oil, either for cooking or for frying, and other risk factors.

DISCUSSION

In this case-control study in India, we found inverse associations between IHD risk and consumption of vegetables, specifically green leafy vegetables, and use of mustard oil. Risk steadily

TABLE 2
Distribution of ischemic heart disease (IHD) risk factors according to total vegetable intake (not including potatoes) among control subjects in New Delhi and Bangalore, India ($n = 700$)

	Category				
	≤1 serving/d	1–2 servings/d	2–3 servings/d	≥3 servings/d	P for trend
Median (servings/d)	0.8	1.5	2.4	3.5	—
No. of controls	137	333	174	56	—
Age (y)	52.0 ± 11.1[1]	52.0 ± 10.9	50.7 ± 11.6	51.3 ± 9.5	0.7
BMI (kg/m^2)	22.3 ± 4.2	23.3 ± 3.9	23.8 ± 4.2	23.8 ± 3.5	0.004
Waist-to-hip ratio	0.94 ± 0.07	0.94 ± 0.07	0.94 ± 0.07	0.93 ± 0.07	0.2
Alcohol intake (servings/d)	0.10 ± 0.55	0.15 ± 0.6	0.16 ± 0.78	0.32 ± 1.3	0.2
Exercise (MET-min/d)[2]	90.2 ± 144.0	98.0 ± 179.1	100.5 ± 142.4	117.3 ± 146.5	0.3
Household income (rupees/mo)	6268 ± 11 695	9099 ± 13 566	9019 ± 6346	13711 ± 16 139	0.02
Female (%)	12.4	12.0	13.2	3.6	0.7
Current cigarette smokers (%)	8.8	15.0	11.5	19.6	0.1
Current bidi smokers (%)	16.1	12.9	5.7	8.9	0.01
Family history of IHD (%)	28.5	33.3	29.9	28.6	1.0
History of hypertension (%)	15.3	14.7	19.5	17.9	0.6
History of high cholesterol (%)	0	0.3	1.7	0	< 0.0001
History of diabetes (%)	14.6	11.1	14.9	14.3	0.04
≥12 y of schooling (%)	21.2	45.4	52.3	66.1	< 0.0001
Hindu religion (%)	80.3	83.2	88.0	83.9	0.03

[1] $\bar{x}$ ± SD (all such values).
[2] MET, metabolic equivalent.

TABLE 3
Relative risk (RR) of ischemic heart disease (IHD) according to potential risk factors[1]

	No. of cases	No. of controls	Age- and sex-adjusted RR (95% CI)[2]	Multivariate RR (95% CI)[3]
	n	*n*		
Cigarette smoking				
Never smoker	120	436	1.0	1.0
>10 cigarettes/d	61	28	7.6 (4.5, 12.8)	7.5 (4.0, 14.0)
Bidi smoking				
Never smoker	120	436	1.0	1.0
>10 bidis/d	71	39	8.0 (4.8, 13.3)	6.7 (3.7, 12.3)
BMI (kg/m^2)				
<20	49	151	1.0	1.0
≥25	121	215	2.5 (1.6, 3.9)	2.6 (1.4, 4.7)
Waist-to-hip ratio				
≤0.9	60	172	1.0	1.0
>1.0	75	104	3.2 (1.9, 5.2)	2.5 (1.3, 4.8)
Leisure-time exercise				
Nonexerciser	189	339	1.0	1.0
≥145 MET-min/d	51	189	0.45 (0.3, 0.7)	0.44 (0.3, 0.7)
Family history of IHD				
No	189	470	1.0	1.0
Yes	146	218	1.9 (1.4, 2.5)	2.3 (1.6, 3.5)
History of hypertension				
No	195	506	1.0	1.0
Yes	106	114	2.6 (1.9, 3.7)	2.2 (1.5, 3.5)
History of high cholesterol				
No	202	477	1.0	1.0
Yes	9	4	6.3 (1.9, 21.0)	4.5 (0.7, 27.7)
History of diabetes				
No	240	519	1.0	1.0
Yes	61	91	1.5 (1.0, 2.2)	1.3 (0.8, 2.2)
Education				
Highest level	17	59	1.0	1.0
None	42	84	2.1 (1.1, 4.1)	2.5 (1.0, 6.0)
Household income				
>10 000 rupees/mo	69	161	1.0	1.0
<3000 rupees/mo	107	161	1.6 (1.1, 2.5)	1.6 (0.9, 3.0)
Hindu religion				
No	75	113	1.0	1.0
Yes	275	587	0.7 (0.5, 1.0)	0.9 (0.6, 1.4)

[1] MET, metabolic equivalent. RR estimates were obtained by using conditional logistic regression analysis controlled for the matching factors (age, sex, and hospital) and then additional potential risk factors.

[2] Also adjusted for hospital.

[3] Covariates controlled for in the multivariate model were as follows: age; sex; hospital; cigarette smoking [never, current (≤10 cigarettes/d, >10 cigarettes/d)]; bidi smoking [never, current (≤10 bidis/d, >10 bidis/d)]; BMI, in kg/m^2 (<20, ≥20–22.5, ≥22.5–25, ≥25); waist-to-hip ratio (≤0.9, >0.9–0.95, >0.95–1.0, >1.0); leisure time physical exercise (none, <145 MET-min/d, ≥145 MET-min/d); history of hypertension (no, yes); history of diabetes (no, yes); history of high cholesterol (no, yes); family history of IHD (no, yes); alcohol intake (no intake, any intake); education (none, primary school, middle, secondary, higher secondary, college, graduate or professional); household income (<3000, 3000–6000, 6000–10 000, >10 000 rupees/mo); and Hindu religion (no, yes).

declined across quartiles of intake of green leafy vegetables: intake of ≥ 3 servings/wk was associated with a threefold lower risk than was intake of < 1 serving/wk. Use of mustard oil, which is rich in α-linolenic acid, was associated with a twofold lower risk than was use of sunflower or other oils. Alternatively, the addition of vanaspati, which is rich in *trans* fatty acids, to food was associated with a moderate increase in IHD risk, although there was no significant association with vanaspati used for cooking or frying.

Potential sources of bias in the present study include the selection of controls and a differential recall among cases and controls. To address selection bias, we obtained controls from 7 different outpatient clinics and inpatient wards. Thus, if an association existed between the exposure of interest and the disease status of one control group, the bias that may have resulted would have been diluted (21). Although health-conscious individuals may have been more likely to give consent for the study, study participation was high (≈99%), so bias from this source is unlikely. Controls in our population were slightly more educated than were the cases but had lower incomes. Controlling for these socioeconomic status factors did not alter our findings. Differential recall of dietary intake is also a potential concern, but it seems unlikely to explain the association with mustard oil in this study, because the use of mustard oil is not perceived as particularly healthy or unhealthy.

Because a companion nutrient database was not available for either FFQ, total energy intake could not be computed. Although

TABLE 4
Relative risk (RR) of ischemic heart disease (IHD) by intake of food groups[1]

	Median	No. of cases	No. of controls	Relative risk (95% CI): Age- and sex-adjusted RR[2]	Age-, sex-, and smoking-adjusted RR[2]	Age-, sex-, and nondietary covariate–adjusted RR[3]	Age-, sex-, nondietary, and dietary covariate–adjusted RR[4]
		n	*n*				
Total vegetables (not potatoes)							
≤1 servings/d	0.8	79	137	1.0	1.0	1.0	1.0
1–2 servings/d	1.5	168	333	0.85 (0.60, 1.21)	0.81 (0.54, 1.21)	0.73 (0.45, 1.19)	0.73 (0.44, 1.20)
2–3 servings/d	2.4	83	174	0.79 (0.53, 1.19)	0.78 (0.49, 1.25)	0.63 (0.35, 1.22)	0.62 (0.34, 1.12)
>3 servings/d	3.5	20	56	0.59 (0.32, 1.08)	0.36 (0.18, 0.73)	0.27 (0.11, 0.64)	0.33 (0.13, 0.82)
P for trend				0.09	0.01	0.006	0.006
Green leafy vegetables							
≤1 servings/wk	0.5	91	175	1.0	1.0	1.0	1.0
1–2 servings/wk	1.0	133	209	1.12 (0.75, 1.66)	0.93 (0.59, 1.50)	0.83 (0.47, 1.44)	0.85 (0.48, 1.52)
2–3 servings/wk	2.0	72	161	0.78 (0.49, 1.23)	0.71 (0.45, 1.26)	0.60 (0.32, 1.14)	0.55 (0.28, 1.06)
>3 servings/wk	3.5	54	155	0.61 (0.37, 0.98)	0.43 (0.24, 0.75)	0.33 (0.17, 0.64)	0.34 (0.17, 0.69)
P for trend				0.002	0.0002	0.0001	0.0002
Potatoes							
≤1 servings/wk	0.2	105	204	1.0	1.0	1.0	1.0
1–2 servings/wk	1	62	151	0.81 (0.55, 1.19)	0.82 (0.53, 1.27)	1.05 (0.64, 1.71)	1.08 (0.64, 1.80)
2–4 servings/wk	3	98	175	1.15 (0.77, 1.72)	1.27 (0.80, 2.00)	1.37 (0.79, 2.35)	1.47 (0.83, 2.60)
>4 servings/wk	7	85	170	1.06 (0.65, 1.71)	0.87 (0.50, 1.51)	0.75 (0.39, 1.45)	0.86 (0.42, 1.75)
P for trend				0.7	0.8	0.3	0.7
Fruit							
≤1 serving/d	0.5	145	304	1.0	1.0	1.0	1.0
1–2 servings/d	1.4	128	243	1.10 (0.82, 1.48)	1.16 (0.82, 1.62)	1.26 (0.84, 1.91)	1.45 (0.93, 2.26)
2–3 servings/d	2.4	43	104	0.88 (0.58, 1.33)	1.15 (0.72, 1.83)	1.21 (0.68, 2.14)	1.29 (0.71, 2.35)
>3 servings/d	3.6	34	49	1.46 (0.89, 2.39)	1.96 (1.11, 3.46)	2.11 (1.03, 4.32)	2.46 (1.15, 5.25)
P for trend				0.4	0.04	0.06	0.03
Cereals							
≤5 servings/d	4.3	78	113	1.0	1.0	1.0	1.0
5–7 servings/d	6.0	101	223	0.65 (0.45, 0.94)	0.58 (0.38, 0.89)	0.71 (0.48, 1.16)	0.76 (0.46, 1.25)
7–9 servings/d	8.0	84	172	0.68 (0.45, 1.02)	0.61 (0.38, 0.97)	0.59 (0.34, 1.01)	0.67 (0.38, 1.17)
>9 servings/d	10.9	87	192	0.61 (0.40, 0.94)	0.49 (0.30, 0.80)	0.49 (0.27, 0.89)	0.47 (0.25, 0.87)
P for trend				0.07	0.02	0.02	0.02
Beans							
≤1 servings/d	0.6	84	146	1.0	1.0	1.0	1.0
1–2 servings/d	1.3	208	432	0.82 (0.59, 1.14)	0.81 (0.55, 1.18)	0.84 (0.54, 1.32)	0.77 (0.48, 1.25)
2–3 servings/d	2.2	44	99	0.74 (0.46, 1.20)	0.74 (0.42, 1.30)	1.02 (0.52, 1.97)	1.05 (0.52, 2.12)
>3 servings/d	3.2	14	23	1.04 (0.50, 2.15)	1.64 (0.70, 3.84)	1.77 (0.65, 4.83)	1.79 (0.62, 5.13)
P for trend				0.5	0.9	0.4	0.3
Dairy							
≤0.5 servings/d	0	100	171	1.0	1.0	1.0	1.0
0.5–1 servings/d	0.8	52	82	1.10 (0.71, 1.69)	0.99 (0.60, 1.65)	1.16 (0.64, 2.10)	1.09 (0.59, 2.04)
1–2 servings/d	1.3	107	223	0.81 (0.58, 1.14)	0.86 (0.58, 1.27)	1.14 (0.71, 1.85)	1.18 (0.71, 1.97)
>2 servings/d	2.4	91	224	0.69 (0.48, 0.98)	0.77 (0.51, 1.17)	1.13 (0.68, 1.89)	1.16 (0.66, 2.02)
P for trend				0.02	0.2	0.7	0.6
Eggs							
0/wk	0	154	319	1.0	1.0	1.0	1.0
>0–1/wk	0.5	93	180	1.05 (0.76, 1.46)	0.93 (0.64, 1.35)	0.82 (0.53, 1.27)	0.77 (0.48, 1.23)
1–2/wk	2.0	46	122	0.79 (0.53, 1.17)	0.74 (0.48, 1.14)	0.51 (0.30, 0.86)	0.50 (0.29, 0.88)
>2/wk	3.6	57	79	1.52 (1.01, 2.27)	1.1 (0.69, 1.77)	0.84 (0.47, 1.49)	0.89 (0.49, 1.62)
P for trend				0.2	0.9	0.5	0.3
Meat (including chicken)							
0 servings/mo	0	153	335	1.0	1.0	1.0	1.0
>0–3 servings/mo	2	67	139	1.08 (0.76, 1.53)	1.03 (0.69, 1.53)	0.85 (0.53, 1.36)	0.83 (0.51, 1.37)
3–6 servings/mo	4	52	93	1.23 (0.83, 1.83)	1.10 (0.70, 1.71)	0.80 (0.47, 1.35)	0.92 (0.54, 1.59)
>6 servings/mo	10	78	133	1.29 (0.92, 1.82)	0.93 (0.62, 1.39)	0.50 (0.30, 0.84)	0.57 (0.33, 0.98)
P for trend				0.13	0.7	0.009	0.06

(*Continued*)

TABLE 4 (*Continued*)

	Median	No. of cases	No. of controls	Relative risk (95% CI): Age- and sex-adjusted RR[2]	Age-, sex-, and smoking-adjusted RR[2]	Age-, sex-, and nondietary covariate–adjusted RR[3]	Age-, sex-, nondietary, and dietary covariate–adjusted RR[4]
Fish							
No intake	0	233	486	1.0	1.0	1.0	1.0
Any intake, servings/d	0.07	117	214	1.15 (0.87, 1.52)	0.83 (0.70, 1.34)	0.69 (0.46, 1.03)	0.72 (0.47, 1.09)

[1] $n = 1050$. MET, metabolic equivalent. RR estimates were obtained by using conditional logistic regression analysis controlled for the matching factors (age, sex, and hospital) and then additional potential risk factors.

[2] Also adjusted for hospital.

[3] Covariates controlled for in the multivariate model were as follows: age; sex; hospital; cigarette smoking [never, past, current (≤2, >2–6, >6–12.5, >12.5 cigarettes/d)]; bidi smoking [never, past, current (≤5.5, >5.5–10, >10–20, >20 bidis/d)]; BMI, in kg/m^2 (<20, ≥20–22.5, ≥22.5–25, ≥25); waist-to-hip ratio (≤0.9, >0.9–0.95, >0.95–1.0, >1.0); leisure time physical exercise (none, <145 MET-min/d, ≥145 MET-min/d); history of hypertension (no, yes); history of diabetes (no, yes); history of high cholesterol (no, yes); family history of IHD (no, yes); alcohol intake (no intake, any intake); education (none, primary school, middle, secondary, higher secondary, college, graduate or professional); household income (<3000, 3000–6000, 6000–10 000, >10 000 rupees/mo); and Hindu religion (no, yes).

[4] Additional dietary covariates controlled for were as follows: cereal intake (≤5, 5–7, 7–9, >9 servings/d, except in cereal analysis), green leafy vegetable intake (≤1, 1–2, 2–3, ≥3 servings/wk, except in total vegetable and green leafy vegetable analyses), mustard oil use in cooking (no, yes), and mustard oil use in frying (no, yes).

the observed associations may be confounded by total energy intake, further adjustment for total cereal intake, a large percentage of total energy intake in the Indian diet, did not alter our findings for green leafy vegetables and use of mustard oil. Control for physical activity, a measure of energy expenditure, also did not change our results. However, the inverse association with cereal intake and IHD risk needs to be interpreted cautiously because it may represent an association with total energy intake resulting from a protective effect of higher physical activity.

TABLE 5
Relative risk (RR) of ischemic heart disease (IHD) by intake of beverages[1]

	Median	No. of cases	No. of controls	Relative risk (95% CI): Age- and sex-adjusted RR[2]	Age-, sex- and smoking-adjusted RR[2]	Age-, sex-, and nondietary covariate–adjusted RR[3]	Age-, sex-, nondietary, and dietary covariate–adjusted RR[4]
		n	*n*				
Tea							
≤1 cup/d	0.03	103	252	1.0	1.0	1.0	1.0
1–2 cups/d	2	87	202	1.12 (0.78, 1.62)	1.66 (0.73, 1.66)	1.13 (0.70, 1.81)	1.16 (0.71, 1.91)
2–3 cups/d	3	51	132	1.04 (0.68, 1.61)	0.83 (0.52, 1.33)	0.85 (0.50, 1.45)	0.81 (0.45, 1.43)
>3 cups/d	5	109	114	2.91 (1.93, 4.39)	1.74 (1.08, 2.8)	1.63 (0.95, 2.80)	1.71 (0.96, 3.03)
P for trend				<0.0001	0.05	0.1	0.1
Coffee							
0 cups/d	0	187	393	1.0	1.0	1.0	1.0
>0–1 cups/d	0.3	84	164	1.13 (0.8, 1.59)	1.34 (0.9, 2.01)	1.72 (1.07, 2.76)	1.93 (1.17, 3.17)
1–2 cups/d	2	41	78	1.19 (0.74, 1.92)	1.38 (0.8, 2.36)	1.60 (0.85, 3.00)	1.70 (0.90, 3.24)
>2 cups/d	3.5	38	65	1.33 (0.81, 2.19)	1.31 (0.74, 2.33)	1.67 (0.86, 3.24)	1.64 (0.82, 3.26)
P for trend				0.3	0.4	0.3	0.3
Alcohol							
No intake	0	238	559	1.0	1.0	1.0	1.0
Any intake, servings/d	0.29	112	141	2.02 (1.47, 2.77)	1.23 (0.83, 1.82)	1.19 (0.76, 1.88)	1.18 (0.73, 1.92)

[1] 1 cup = 240 mL. RR estimates were obtained by using conditional logistic regression analysis controlled for the matching factors (age, sex, and hospital) and then additional potential risk factors.

[2] Also adjusted for hospital.

[3] Covariates controlled for in the multivariate model were as follows: age, sex, hospital, smoking, BMI, waist-to-hip ratio, physical activity, history of hypertension, history of diabetes, history of high cholesterol, family history of IHD, alcohol intake, education, household income, and Hindu religion (*see* Table 4 for categories).

[4] Additional dietary covariates controlled for were as follows: cereal intake, green leafy vegetable intake, mustard oil use in cooking, and mustard oil use in frying (*see* Table 4 for categories).

Our findings are consistent with the results of epidemiologic research conducted in Western countries, which suggested protective effects of vegetables, green leafy vegetables, and cereals against IHD risk. Recent prospective findings from nearly 40 000 US women showed that, compared with those consuming 1.5 servings of vegetables/d, women consuming 6.8 servings were at 55% lower risk of total cardiovascular disease (22). In a prospective study among US men, persons consuming 5-7 fruit and vegetable servings/d had an RR of 0.73 for MI, compared with those eating < 3 servings/d (23). Lower coronary mortality has also been observed with high levels of vegetable and fruit consumption in Finland (24, 25). Case-control findings from Italy suggest significant protective effects of vegetable consumption on IHD risk (26, 27). The elevation in risk observed with fruit intake in the multivariate analysis in the present study (after additional control for dietary covariates) is inconsistent with the inverse associations observed with vegetables and green leafy vegetables.

The inverse association that we observed between mustard oil consumption and IHD risk may support a beneficial effect of α-linolenic acid intake. α-Linolenic acid reduces the adhesion-aggregation tendency of blood platelets, which should decrease the risk of thrombosis and consequent MI (28, 29). A significant inverse association of linolenic acid intake with risk of IHD was reported in several prospective studies, including the Multiple Risk Factor Intervention Trial (16), the Health Professionals' Follow-up Study (17), the Alpha-Tocopherol Beta-Carotene Cancer Prevention Study (13), and the Nurses' Health Study (18). The inverse association was restricted to fatal IHD in the Alpha-Tocopherol Beta-Carotene Cancer Prevention Study and the Nurses' Health Study but was also observed for nonfatal IHD in the Health Professionals' Follow-up Study. The inverse association between use of mustard oil and risk of IHD in our study may perhaps be explained by the low fish intake in New Delhi and Bangalore; only 32% of the study population consumed fish (median intake of 0.07 servings/d). In populations with low fish consumption, α-linolenic is the main source of essential $n-3$ polyunsaturated fatty acids. Unlike canola oil, mustard oil is also a source of erucic acid (22:1), a long-chain monounsaturated fatty acid. It has been suggested that erucic acid in mustard oil may counterbalance the beneficial effects of linolenic acid by increasing serum LDL-cholesterol and triacylglycerol concentrations (30). However, our findings suggest that the overall effect of mustard seed oil is beneficial. Unlike the higher erucic acid contents of rapeseed grown in India, most rapeseed grown in Europe and North America is bred to contain low amounts of erucic acid ($\leq$ 5%; 31).

Trials have assessed the effects of vegetable intake on IHD risk. The Indo-Mediterranean Diet Heart Study, a randomized trial of persons with preexisting heart conditions in India, compared the effects of a Mediterranean-style diet rich in α-linolenic acid (including fruit, vegetables, nuts, whole grains, and mustard seed or soybean oils) on the recurrence of cardiac events with those of a National Cholesterol Education Program Step I prudent diet (32). After 2 y of follow-up, those in the Indo-Mediterranean group had a significant reduction in cardiac endpoints compared with controls, including a 53% lower risk of nonfatal MI, a 67% lower risk of sudden cardiac death, and a 52% lower risk of total cardiac endpoints. The Indian Heart Study, a randomized controlled trial of IHD patients, advised patients in

TABLE 6

Relative risk (RR) of ischemic heart disease (IHD) by intake of discretionary (added) oils and fats[1]

	Median	No. of cases	No. of controls	Relative risk (95% CI): Age- and sex-adjusted RR[2]	Age-, sex-, and smoking-adjusted RR[2]	Age-, sex-, and nondietary covariate–adjusted RR[3]	Age-, sex-, nondietary, and dietary covariate–adjusted RR[4]
Vegetable oil		*n*	*n*				
≤1 small teaspoons added/d	0	191	323	1.0	1.0	1.0	1.0
1–2/d	1.3	105	262	0.60 (0.43, 0.83)	0.60 (0.41, 0.88)	0.61 (0.39, 0.94)	0.59 (0.37, 0.94)
2–3/d	2.1	30	79	0.58 (0.35, 0.96)	0.54 (0.31, 0.97)	0.70 (0.36, 1.39)	0.71 (0.35, 1.46)
>3/d	4	24	36	1.10 (0.60, 2.0)	1.10 (0.54, 2.25)	1.23 (0.54, 2.78)	1.55 (0.65, 3.73)
P for trend				0.2	0.2	0.5	0.8
Vanaspati							
Nonuse	0	300	632	1.0	1.0	1.0	1.0
Use, small teaspoons added/d	1.3	50	68	1.70 (1.10, 2.64)	1.63 (0.98, 2.72)	1.81 (0.99, 3.31)	1.31 (0.68, 2.51)
Ghee							
0 small teaspoons added/d	0	175	345	1.0	1.0	1.0	1.0
>0–2/d	0.6	79	131	1.22 (0.86, 1.72)	1.40 (0.94, 2.07)	1.66 (1.03, 2.68)	1.69 (1.02, 2.8)
2–6/d	4	51	98	0.98 (0.66, 1.46)	1.03 (0.65, 1.62)	1.27 (0.75, 2.14)	1.37 (0.79, 2.40)
>6/d	8.4	45	126	0.62 (0.40, 0.96)	0.68 (0.41, 1.12)	0.94 (0.52, 1.71)	1.15 (0.59, 2.22)
P for trend				0.03	0.1	0.7	0.7

[1] 1 teaspoon = 5 mL. RR estimates were obtained by using conditional logistic regression analysis controlled for the matching factors (age, sex, and hospital) and then additional potential risk factors.

[2] Also adjusted for hospital.

[3] Covariates controlled for in the multivariate model were as follows: age, sex, hospital, smoking, BMI, waist-to-hip ratio, physical activity, history of hypertension, history of diabetes, history of high cholesterol, family history of IHD, alcohol intake, education, household income, and Hindu religion (*see* Table 4 for categories).

[4] Additional dietary covariates controlled for were as follows: cereal intake, green leafy vegetable intake, mustard oil use in cooking, and mustard oil use in frying (*see* Table 4 for categories).

TABLE 7
Relative risk (RR) of ischemic heart disease (IHD) according to fats and oils used in cooking and frying[1]

	No. of cases	No. of controls	Relative risk (95% CI)			
			Age- and sex-adjusted RR[2]	Age-, sex,- and smoking-adjusted RR[2]	Age-, sex-, and nondietary covariate–adjusted RR[3]	Age-, sex-, nondietary, and dietary covariate–adjusted RR[4]
	n	*n*				
Cooking						
Sunflower oil (reference)	111	233	1.0	1.0	1.0	1.0
Ghee	11	32	0.67 (0.31, 1.47) [0.3][5]	0.59 (0.23, 1.51) [0.30]	0.48 (0.16, 1.45) [0.2]	0.52 (0.17, 1.55) [0.2]
Vanaspati	35	51	1.16 (0.65, 2.08) [0.6]	0.91 (0.46, 1.80) [0.8]	0.75 (0.33, 1.74) [0.5]	0.67 (0.29, 1.61) [0.4]
Mustard oil	54	169	0.55 (0.33, 0.91) [0.02]	0.44 (0.25, 0.79) [0.006]	0.49 (0.24, 0.99) [0.05]	0.46 (0.22, 0.95) [0.04]
Peanut oil	72	132	1.18 (0.81, 1.71) [0.4]	1.21 (0.78, 1.86) [0.4]	1.20 (0.73, 1.96) [0.5]	1.19 (0.72, 1.97) [0.5]
Safflower oil	14	14	1.92 (0.85, 4.37) [0.1]	2.06 (0.83, 5.11) [0.1]	1.45 (0.52, 4.08) [0.5]	1.27 (0.43, 3.73) [0.7]
Frying						
Sunflower oil (reference)	113	233	1.0	1.0	1.0	1.0
Ghee	9	24	0.69 (0.28, 1.70) [0.4]	0.62 (0.22, 1.72) [0.4]	0.55 (0.17, 1.83) [0.3]	0.62 (0.19, 1.99) [0.4]
Vanaspati	53	87	1.02 (0.60, 1.75) [0.9]	0.91 (0.49, 1.68) [0.8]	0.74 (0.33, 1.64) [0.5]	0.66 (0.29, 1.49) [0.3]
Mustard oil	31	130	0.39 (0.22, 0.68) [0.001]	0.31 (0.16, 0.59) [0.0003]	0.29 (0.13, 0.64) [0.002]	0.25 (0.11, 0.57) [0.001]
Peanut oil	75	139	1.17 (0.81, 1.69) [0.4]	1.21 (0.79, 1.84) [0.4]	1.18 (0.73, 1.89) [0.5]	1.17 (0.72, 1.90) [0.5]
Safflower oil	10	18	1.03 (0.44, 2.44) [0.9]	1.24 (0.47, 3.24) [0.7]	0.88 (0.29, 2.67) [0.8]	0.73 (0.23, 2.34) [0.6]

[1] RR estimates were obtained by using conditional logistic regression analysis controlled for the matching factors (age, sex, and hospital) and then additional potential risk factors.

[2] Also adjusted for hospital.

[3] Covariates controlled for in the multivariate model were as follows: age, sex, hospital, cigarette smoking, bidi smoking, BMI, waist-to-hip ratio, physical activity, history of hypertension, history of diabetes, history of high cholesterol, family history of IHD, alcohol intake, education, household income, and Hindu religion (*see* Table 4 for categories).

[4] Additional dietary covariates controlled for were as follows: cereal intake (≤5.5, 5–7, 7–9, >9 servings/d) and green leafy vegetable intake (≤0.7, 0.7–1.05, 1.05–2.1, >2.1 servings/wk).

[5] *P* value in brackets (all such values). *P* values < 0.01 should be considered statistically significant because of multiple comparisons.

the experimental group to eat a diet rich in fruit, vegetables, whole grains, and nuts rich in α-linolenic acid (33). The control group did not receive dietary recommendations, yet all subjects were advised on dietary fat reduction, smoking, alcohol intake, stress management, and exercise. At the 1-y follow-up, the experimental group showed a 38% reduction in nonfatal MI and a 32% reduction in fatal MI. The Indian Experiment of Infarct Survival Study assessed the effects of treatment with fish oil and mustard oil, both of which are high in n−3 fatty acids, among patients with suspected acute MI (34). After 1 y, total cardiac events were significantly lower in the fish oil (24.5%) and mustard oil (28%) groups than in the placebo group (34.7%). The Lyon Diet Heart Study, a secondary prevention trial among patients with a first MI, promoted a diet rich in α-linolenic acid, with higher intakes of bread, fruit and vegetables, fish, and rapeseed (canola) oil and less meat, butter, and cream (35). After 27 mo of follow-up, risk of fatal and nonfatal MI was reduced to 0.27 (95% CI: 0.12, 0.59).

The inverse association that we observed between intake of green leafy vegetables and risk of IHD could be explained by the protective effects of folate. Low folic acid intake is associated with increased plasma homocysteine concentrations and elevated risk of IHD (8, 36-39). Elevated homocysteine concentrations may contribute to the higher IHD rates among Asian Indians living abroad than among Europeans (6). In that study, the authors attributed the elevated homocysteine concentrations in Indians to low concentrations of folate and vitamin B-12.

The somewhat elevated risk we observed with consumption of vanaspati, which is high in *trans* fatty acids, may be attributed in part to the effects of *trans* fatty acids on blood lipids. Dietary *trans* fatty acids raise LDL, triacylglycerols, and lipoprotein(a); lower HDL cholesterol; and interfere with essential fatty acid metabolism (12, 40-42).

Our findings highlight the importance of diets rich in vegetables and α-linolenic acid in the prevention of IHD. However, more studies, including prospective investigations, are required to examine these associations further in India. Given the limited resources in India, primary prevention is critical in addressing the ongoing IHD epidemic. Public health programs should focus on lifestyle habits, including smoking cessation, improvements in physical activity, and dietary modification.

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APPENDIX A

Foods included on the Indian food-frequency questionnaire[1]

Total vegetables (no potatoes)	Fruit	Cereals	Beans	Dairy	Meat	Fish	Mixed dishes	Snacks and desserts	Alcohol
Cauliflower	Apple	Rice	Dal (lentils)	Toned milk	Goat or lamb curry	Fish curry	Creamed soup	Nuts	Beer
Capsicum	Citrus	Fried rice	Chickpea	Skimmed milk	Other goat or lamb	Other fish	Noncreamed soup	Sweet biscuit	Wine
French beans	Banana	White bread	Dried beans	Whole milk	Chicken curry	Deep-fried fish	Vegetarian pizza	Cream biscuit	Liquor
Broad beans	Fruit salad	Bread roll	Sambar	Flavored milk	Chicken kabab	Seafood	Nonvegetarian pizza	Samosa	
Peas	Grapes	Roti	Rasam	Milkshake	Fried chicken		Vegetable sandwich	Pakora	
Okra	Berries	Missi roti		Lassi	Beef curry		Pao baji	Vegetable cutlet	
Green leafy vegetables	Plum	Naan		Yogurt	Other beef		Masala dosa	French fries	
Salad (fresh mixed)	Mango	Paratha		Raita (yogurt dish)	Ham, salami, bacon			Namkeen	
Tomato	Watermelon	Puri		Cheese	Sausage			Chaat	
Radish	Papaya	Bhatura		Paneer	Liver			Crisp snack (chips)	
Carrots, raw	Sapota	Upma							
Carrots, cooked	Guava	Stuffed paratha							
Pumpkin	Lichi	Cereals (breakfast)							
Gourd	Pomegranate	Cooked cereals							
Parmal	Custard apple	Uttapam							
Corn	Dried fruit	Dokla							
Mushroom		Idli							
Kathal		Biryani							
Drumstick		Vada							
Vegetable kofta		Lemon rice							
Vegetable kurma (mixed vegetables		Bisibele bath							
		Poha (rice flakes)							
		Ragiball							
		Vegetable sandwich							
		Vegetable noodles							
		Nonvegetable noodles							

[1] The following foods were assessed in separate groups: green leafy vegetables, potatoes (including yams), eggs, tea, and coffee.